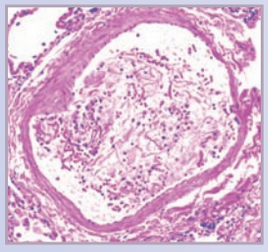

AMNIOTIC FLUID EMBOLISM

Abstract
Amniotic fluid embolism (AFE) is a rare but serious and potentially deadly complication of pregnancy that is unpreventable and unpredictable. Most AFE events occur during labor; however, approximately one third happen during the immediate postpartum period. Presentation is abrupt and thought to be an abnormal response to fetal materials entering maternal circulation through the placental insertion site. Care providers must recognize the signs and symptoms of AFE and react quickly to treat potential complications. This can be challenging as there are no set diagnostic criteria or specific laboratory tests. Generally, the diagnosis is based on clinical status when the classic triad of hypoxia, hypotension, and subsequent coagulopathy are noted in a laboring woman or woman who just gave birth, and no other plausible explanation can be determined. Proper treatment of AFE requires a multidisciplinary approach to decrease maternal morbidity and mortality. Knowledge, simulation, and familiarization of a Massive Obstetric Transfusion protocol can help all members of the perinatal team recognize and respond to women with AFE in a timely and effective manner. A case study is presented of a woman with a seemingly normal obstetric course that became complicated rapidly following development of an AFE.
Key Words: Amniotic fluid embolism; Complication of labor and birth; Disseminated intravascular coagulopathy; Postpartum hemorrhage.

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The first case of amniotic fluid embolism (AFE) was documented in 1926 in a Brazilian medical journal following death of a woman with an intrauterine fetal demise and sepsis. After birth of the fetus, the woman developed sudden cardiac arrest. Fetal cells were discovered in the maternal pulmonary vasculature during an autopsy (Dobbenga-Rhodes, 2009). In 1941, Steiner and Luschbaugh added a more detailed description of the clinical manifestations of AFE including sudden shock and pulmonary edema during labor (Dobbenga-Rhodes). Despite an incomplete understanding of AFE, research over last 20 years has changed previous concepts of its causation and pathophysiology (Clark, 2010). An abnormal immune response, in addition to mechanical blockage of maternal pulmonary vasculature with components of amniotic fluid, are likely leading causative factors (Clark, 2014).

Initially, AFE had a mortality rate of 80% to 90%; however, with improvements in critical care, recent reported mortality rates range from 11% to 60% (Clark, 2014; Oi et al., 2014; Price, 2012). Exact incidence is unknown due to inadequate reporting and lack of consistent diagnostic criteria (Society for Maternal-Fetal Medicine [SMFM], Pacheco, Saade, Hankins, & Clark, 2016); however, experts estimate an AFE incidence of between 1 in 12,500 and 1 in 50,000 births (Clark, 2014; Conde-Agudelo & Romero, 2009; Rath, Hoferr, & Sinicina, 2014). Mortality rates and incidence may be underestimated due to inconsistencies with diagnosis and death certificate reporting (Clark; Rath et al.). Amniotic fluid embolism events usually occur during labor and birth, but may also happen during the immediate postpartum period and during or after termination of pregnancy (Price). Approximately 56% of women do not survive the first 2 hours following the acute event (Rath et al.). The Centers for Disease Control and Prevention (2016) lists AFE as the 9th leading cause of pregnancy-related deaths in the United States and responsible for 5.7% of maternal deaths.

Definition

Amniotic fluid embolism is a rare, life-threatening complication of pregnancy that is unpreventable and unpredictable (Kramer, Abenhaim, Dahhou, Rouleau, & Berg, 2013; Price, 2012). It presents with an abrupt and severe onset following components of amniotic fluid, including minute fetal materials, entering maternal circulation through the placental site causing abnormal activation of proinflammatory mediator systems comparable to the systemic inflammatory response syndrome (SMFM et al., 2016). These materials from the amniotic fluid can include fetal squamous cells, debris, mucin, meconium, or lanugo (Nakagami et al., 2015).

Amniotic fluid embolism is a rare, life-threatening complication of pregnancy.

Pathophysiology

Pathophysiology of AFE remains unknown. There is a growing consensus that clinical manifestations may not primarily be the result of a physical obstruction of pulmonary vessels by fetal material, but rather a maternal immunological response to vasoactive substances and procoagulant substances that cause activation in the endothelium thus resulting in a massive inflammatory reaction (Rath et al., 2014). This mechanism is unclear, but it appears symptoms resemble anaphylaxis. It is usually characterized by sudden dyspnea, cardiopulmonary collapse, and coagulopathy (Kramer et al., 2013). According to a recent clinical guideline from SMFM et al. (2016), the typical presentation of AFE involves a triad of symptoms consisting of hypoxia, hypotension or cardiac arrest, and coagulopathy. Some experts describe the presentation as systemic inflammatory response syndrome or reaction resembling shock, following the entrance of the fetal antigen into maternal bloodstream (Clark, 2014; Schoening, 2006).

Severity of the reaction may depend on how the body responds to the foreign material. The material contains several substances (bradykinins, histamines, cytokines, and procoagulant) that cause an immune response activating the endothelial cells, leading to an inflammatory reaction. Antibody production, chemical signals, and specific enzymes also are thought to contribute to the hemodynamic changes in the body following the entrance of the foreign material (Shen, Wang, Yang, & Chen, 2016). Nakagami et al. (2015) speculate that introduction of materials is related to a disruption in the barrier between the amniotic fluid and maternal circulation. Once the amniotic fluid enters the maternal vasculature through the endocervical veins, uterine lesions, or site of placental attachment, there is an initial pulmonary vasoconstriction causing increased pressure and leading to right-sided heart failure. This cardiopulmonary interruption causes disruption in gas exchange leading to respiratory failure and hypoxia, which results in the wide range of signs and symptoms seen in Table 1. Rath et al. (2014) report that following the initial phase of AFE, the second phase involves left-sided heart failure leading to pulmonary edema. Patients develop severe coagulopathy resulting in life-threatening disseminated intravascular coagulation (DIC). In a normal clotting event, localized cell damage causes the endothelial cells to generate tissue factor. Tissue factor is then responsible for activating coagulation cascade. In AFE, the fetal material containing tissue factor enters the maternal bloodstream leading to generalized endothelial reaction, activating widespread, abnormal coagulation, triggering DIC (Cunningham & Nelson, 2015). See Figure 1 (adapted from Clark, 2014; Schoening, 2006).

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Amniotic fluid embolism presents with an abrupt and severe onset following an abnormal response to fetal materials entering maternal circulation.

Risk Factors

Because incidence of AFE is so rare, risk factors are not distinct. According to the national registry, 70% of AFE cases occur during labor, 11% after vaginal birth, and 19% during cesarean birth (SMFM et al., 2016). Associations have been noted between development of AFE and induction of labor, advanced maternal age, women with multiple pregnancies, cesarean birth, operative vaginal birth, placenta previa, placental abruption, and eclampsia (Oi et al., 2014; Price, 2012). Terminations of pregnancy, periods of raised intra-amniotic pressure, cervical lacerations, uterine rupture, and artificial rupture of membranes are also a risk factors associated with AFE (Oi et al.; Price). Shen et al. (2016) found AFE was 71.7% more common in women pregnant with male infants.

Signs, Symptoms, and Clinical Manifestation

Signs and symptoms of AFE are often divided into two phases, with some patients manifesting precursory symptoms (Table 1). Precursory symptoms of AFE consist of agitation, numbness, feeling cold, lightheadedness, chest pain, panic, distress, nausea, vomiting, and impeding sense of doom (Rath et al., 2014; Shen et al., 2016; SMFM et al., 2016). These prodromal symptoms usually lead to the collapse, or initial phase, of AFE within 2 hours (Shen et al.). Initially, clinical signs and symptoms include: severe abrupt hypotension, arrhythmia, cardiac arrest, respiratory failure, changes in neurologic status, and atonic hemorrhage. The second phase includes involvement of left-ventricular failure, clotting dysfunction leading to DIC, consequent pulmonary edema, and reactive hypovolemia (Price, 2012; SMFM et al.). The symptoms of AFE indicate an acute emergency, but at times may be confused with other complications related to pregnancy. Six case studies also listed DIC or coagulopathy as the sole clinical symptom following birth, although this is an atypical finding (Shen et al.).

Diagnosis

Due to the wide range of signs and symptoms, there is an equally wide range of diagnostic criteria. Several differential diagnoses need to be addressed including, but not limited to: pulmonary embolism, acute myocardial infarction, septic shock, complications related to anesthesia, hypoglycemia, placental abruption, anaphylaxis, eclampsia, and uterine rupture (Price, 2012; Rath et al., 2014). Anytime there is a sudden and rapid deterioration in the maternal cardiovascular system for an unknown reason, AFE should be suspected (SMFM et al., 2016). In the absence of previously mentioned conditions, diagnosis of AFE can be made with at least one of the following: cardiac arrest, hypovolemic shock, respiratory distress, coma, seizure, or DIC (Kramer et al., 2013). Establishing a more definitive diagnosis of AFE in women who do not survive requires an autopsy to determine if there are amniotic fluid components in the maternal pulmonary vasculature (Rath et al.). In women who survive AFE, presence of fetal material is not a reliable diagnostic because fetal cells can be detected in 21% to 100% of pregnant patients without clinical manifestation of AFE (Rath et al.). Currently, there are no definitive laboratory diagnostic criteria. Rath et al. summarize other researched diagnostic markers of AFE including: zinc coproporphyrin, sialyl-Tn Antigen, tryptase, or C3 and C4 complement and detection insulin-like growth factor binding protein-1; however, none have been shown to be consistent in diagnosis. There is no specific recommendation of diagnostic criteria; therefore, diagnosis is based on presenting clinical manifestations and exclusion of other potential diagnoses (Clark, 2014; SMFM et al.).

Treatment

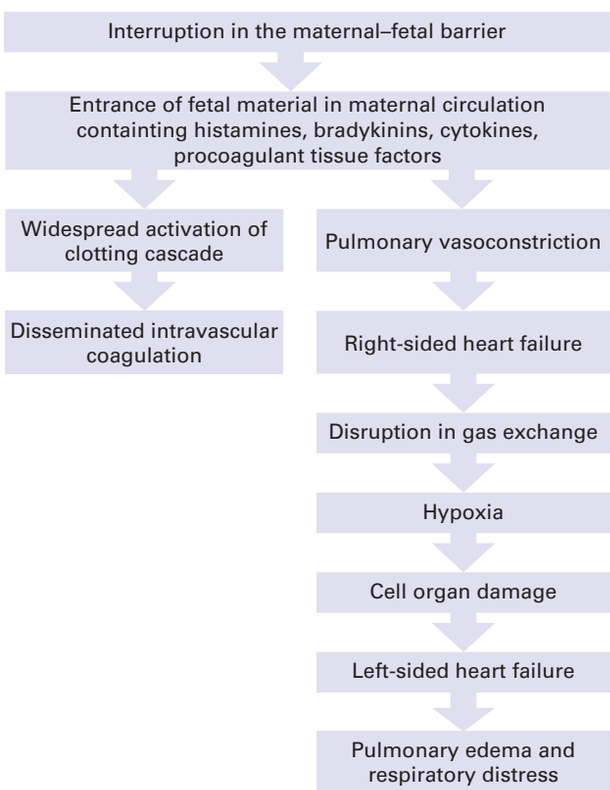
There is no specific treatment protocol unique to AFE due to the variation in clinical presentation. Treatment is geared toward patient symptomology including: correction of cardiovascular collapse, respiratory arrest, coagulopathy, and other interventions to stabilize the woman. Suggestions for immediate supportive treatment based

Table 1. Signs and Symptoms of Amniotic Fluid Embolism

Precursory
Agitation, panic, distress
Numbness, feeling cold, lightheadedness
Chest pain
Nausea and vomiting
Initial phase of AFE
Acute dyspnea and cyanosis (50% to 80%)
Sudden hypotension (56% to 100%)
Cardiac arrest (30% to 80%)
Fetal compromise (20% to 36%)
Seizures/unconsciousness/coma (15% to 50%)
Hemorrhage, life threatening (up to 12%)
Second phase of AFE
Acute left heart failure and pulmonary edema (51% to 100%)
Symptomatic hypovolemia
Myocardial ischemia
Disseminated intravascular coagulation (83%)

Note. Key (%) = percent of women with AFE who develop these signs and symptoms.

Figure 1. Pathophysiology of Amniotic Fluid Embolism



on clinical presentation when AFE is suspected are offered in the recent clinical guidelines from SMFM et al. (2016). High-quality cardiopulmonary resuscitation must be initiated immediately if cardiac arrest develops (SMFM et al.). A multidisciplinary team including anesthesia providers, the critical care team, and maternal–fetal medicine specialists is recommended as best practice for ongoing care (SMFM et al.). In cases where AFE is suspected, ensuring a patent airway with early intubation and sufficient oxygenation are the highest priorities (Rath et al., 2014). Blood for immediate laboratory tests should be drawn to establish coagulation factors, cross matching, and blood gas analysis. Insertion of central venous lines should be quickly considered for monitoring of hemodynamic status. In the event of worsening signs and symptoms, cardiac arrest, or life-threatening arrhythmia, an emergency cesarean should be performed within 3 to 5 minutes (Rath et al.). When blood loss is excessive, a urinary catheter to measure urine output is recommended, as well as, intravenous crystalloid or colloid therapy to replace lost volume. Renal blood flow is extremely sensitive to blood loss; therefore, urine output is preferred at 60 mL per hour but at a minimum should be 30 mL per hour. In the case of a hemorrhage, a Massive Obstetric Transfusion (MOBT) protocol should be initiated (Lyndon, Lagrew, Shields, Main, & Cape, 2015). This type of protocol allows blood products including packed red blood cells (PRBCs), cryoprecipitate, platelets, and fresh frozen plasma (FFP) to be readily available in the blood bank. These protocols are typically started when the patient has already received PRBCs and is still actively bleeding. Massive transfusion protocols, especially when used for obstetric patients, improve patient safety and are thought to reduce overall administration of blood products (Cunningham & Nelson, 2015; Lyndon et al.).

Exemplar Case Presentation

A 22-year-old nulliparous woman at 39 4/7 weeks was admitted in labor at 2330. She progressed well, had epidural anesthesia, and was completely dilated at 0705. After pushing for two and a half hours, vaginal birth of an 8 lb baby boy via low outlet forceps occurred at 0949. A midline episiotomy extended to a 4th degree laceration. Manual exploration of the uterine cavity by the obstetrician following delivery of the placenta found "vastly dilated" uterine vasculature and uterine atony resulting in copious amounts of bright red bleeding. Continuous fundal massage was performed by the nurse. During the repair, the mother complained of her "boobs hurting," which was later identified as chest pain. She became very agitated and asked to rest. Her family remained at the bedside. The woman stated, "I don't feel good," and reported being tired. The circulating nurse noted an increase of bleeding and reported blood covering the width of the bed, completely saturating the peripad, cloth chux, paper chux pad, and dripping to the floor. Vital signs (V/S) were: Blood pressure (BP) ranging from systolic 66 to 72 mmHg

to diastolic 29 to 33 mmHg, heart rate (HR) 120 beats per minute (bpm), respiratory rate 22, and oxygen saturation (SpO_2) 100% (See Table 2 Supplemental Digital Content, http://links.lww.com/MCN/A34). An additional bolus of 30 units of oxytocin in 1,000 mL of intravenous (IV) normal saline (NS) was given as well as methylergonovine maleate 0.2 mg intramuscularly (IM). An indwelling urinary catheter was placed. The fundus was firm with massage at the umbilicus. Within 10 minutes, the fundus was noted to be +2 above the umbilicus with profuse bleeding. New peripads were placed and a pad count was begun. Fundal massage was ongoing. At 1045, laceration repair was completed. With BP of 100/50 mmHg, HR 130 bpm, SpO_2 100% on room air, and decreased vaginal bleeding, the obstetrician estimated blood loss at 1,500 mL and felt the woman was stable enough for the obstetrician to leave the bedside.

At 1100, the obstetrician was notified that the fundus was boggy and there was moderate bleeding. Orders to continue oxytocin 20 units in 1,000 mL NS IV, administer carboprost 250 mcg IM, and begin transfusion of 2 units of PRBCs were received. She began to complain of shortness of breath; V/S were BP 83/37 mmHg, HR 140 bpm, 95% SpO_2. Oxygen was then administered at 10 L/min via nonrebreather face mask, and the certified nurse anesthetist (CRNA) and obstetrician were requested for bedside evaluation. The CRNA administered 10 mg ephedrine IV, started a second peripheral IV, drew blood for laboratory tests, and prepared a fluid warmer for the impending blood transfusion. The mother appeared pale and diaphoretic, but responsive to auditory stimuli. On returning on the unit at 1200, the obstetrician performed a bimanual massage evaluating clots and ordered misoprostol 800 mcg per rectum to assist with uterine tone. The CRNA administered 150 mcg phenylephrine hydrochloride IV to support BP and 2 units of PRBCs were transfused. The obstetrician was again requested to come to bedside to evaluate patient as vaginal bleeding continued. The woman was transferred to the operating room (OR).

Initially in the OR at 1325, BP was 60/34 mmHg. She was preoxygenated and a rapid sequence intubation was performed by anesthesia. During the procedure, the anesthesia team was to unable to obtain BP with an automatic cuff and maternal pulse was weak. She received four 100 mcg boluses of epinephrine IV during the procedure to support BP. A left radial arterial line and a right internal jugular central venous catheter were inserted by the anesthesia team. The first blood gas results at 1345 (See Table 3 Supplemental Digital Content, http://links.lww.com/MCN/A34) showed metabolic acidosis (pH 7.19, HCO3 14.7 mEq/L, 39 CO2 mmHg, base deficit -14) and abnormal coagulation laboratory values including: d-dimer 35.13 µg/mL, fibrinogen (fib) 107.99 mg/dL, thrombin clot time 31.93 seconds, PTT 42.31 seconds, PT 14.49 seconds, INR 1.49, and fibrin split products >20 (<5), revealing DIC. It was also noted that potassium was elevated at 5.9 mEq/L (3.3–5.1).

Proper treatment of AFE requires a multidisciplinary approach to decrease maternal morbidity and mortality.

Repeat labs at 1425 showed severely elevated white blood cells (WBCs) (34.6 x 103/mm3) and a decrease in platelets (PLTs) (91 x 109/L). Schistocytes, anisocytosis, and burr cells were seen, indicating damage to red blood cells (See Table 4 Supplemental Digital Content, http://links.lww.com/MCN/A34). Four ampules of sodium bicarbonate were given by the anesthesia team to correct metabolic acidosis; half of an ampule of calcium chloride to increase myocardial contractility and BP support, and magnesium 2 g to correct electrolytes were administered intravenously. Four additional units of PRBCs and 2 units of FFP were transfused in the OR to combat hypovolemic shock and DIC. The uterus was above the umbilicus and boggy. Bimanual compression was conducted until uterus was firm and methylergonovine maleate 0.2 mg was injected into the lower uterine segment by the obstetrician. The cervix and perineal laceration repair were intact. Curettage and manual exploration of the intrauterine cavity were performed with no significant findings. A uterine tamponade balloon was placed and filled with 480 mL of irrigation. Blood loss was estimated at 1,000 mL during procedure, for a cumulative blood loss of 2,500 mL. During the procedure, BP averaged 103/65 mmHg. At 1545, the intubated patient was transferred to the ICU. On admission to ICU, the HR ranged from 170 to 180 bpm and BP ranged from 90/70 to 80/60 mmHg.

Blood for repeat laboratory tests was drawn and results reviewed. Initial diagnoses were documented as hemorrhagic shock, postpartum hemorrhage, DIC, and possible AFE. The patient's status continued to deteriorate while in the ICU. Abnormal laboratory results were WBC 24.8 x $10^3/mm^3$, hemoglobin (HGB) decreasing to 4.2 g/dL, hematocrit (HCT) 12.7%, and PTLs 72 x

Table 5. Nursing Interventions for Amniotic Fluid Embolism

• Assemble the team necessary to support and resuscitate mother; anesthesia providers at bedside; code team at bedside; initiate cardiopulmonary resuscitation as indicated; if the obstetrician/birth attendant is not at the bedside, notify them to come emergently; other team members as needed
• Obtain crash cart
• Monitor maternal V/S
• If the mother has not given birth, monitor fetal heart rate and uterine activity; notify neonatal team, prepare for cesarean birth and neonatal resuscitation
• Collect specimens for laboratory tests as ordered
• Notify blood bank, complete necessary orders, initiate MOBT, and prepare to administer blood products and coagulation factors
• If the mother has given birth, monitor vaginal blood loss. Pads should be weighed on gram scale (1 g = 1 mL of blood loss)
• Notify gynecology team and other specialties for surgery assistance
• Notify critical care team for patient transfer to critical care unit for invasive hemodynamic monitory
• Support family emotionally by keeping them informed of what is happening and the plan of care

10^9/L. Patient also had elevated lactate level of 4.1 mmol/L (0.9–1.7 mmol/L) indicating prolonged acidosis. Continued vaginal bleeding around uterine tamponade balloon was noted by L&D nurse who remained at the bedside with the critical care team. The obstetrician was contacted and a decision was made to take the patient back for surgery and to initiate the MOBT. The on-call gynecological surgeon was requested to assist with surgery.

At 1743, the sedated and intubated patient was taken back to the OR for an emergent total abdominal hysterectomy. Throughout the case, BP was maintained with a neosynephrine drip. An arterial blood gas was drawn with the following results: pH 7.164, CO_2 45.1 mmHg, HCO_3 16.3 mEq/L, BE -12, reflecting metabolic acidosis. Labs revealed HGB 6.1 g/dL, HCT 18%, PT 12.98 seconds, INR 1.31, fib 371.86 mg/dL, and PTT 32.51 seconds. Two additional ampules of sodium bicarbonate and 1 ampule of calcium gluconate were given IV to maintain appropriate electrolyte imbalance related to the large amount of blood products she received. An additional 6 units of PRBCs, 1 unit of cryoprecipitate, and 1 unit of FFP were transfused during the case. At 1937, the patient was transported back to the ICU, intubated and not requiring BP support.

Upon return to the ICU, a hematology consult was conducted to rule out any clotting disorders; none were found. The patient remained in critical condition, but improved daily. Standing orders were received to keep HGB above 10 g/dL, PLTs greater than 100,000 x 10^9/L, and fibrinogen greater than 150 mg/dL. If these levels fell below set parameters, PRBCs, PLTs, and cryoprecipitate were to be transfused. Over the 24-hour period after birth, she received a total of 11 units of PRBCs, 3 units of FFP, 4 units of PLTs, and 20 units of cryoprecipitate. Laboratory values and arterial gases normalized, the CRNA was able to safely remove the epidural catheter, and she was extubated within 24 hours of placement.

Two days after birth the mother was awake, responsive, and breathing room air. Her indwelling catheter and central line were discontinued and she asked to see her baby. Over the next few days, diet was advanced, the dressing was removed, V/S remained stable, and laboratory values continued to improve. She was discharged 6 days after admission in stable condition.

Clinical Implications

Nurses need to be knowledgeable of signs and symptoms of AFE. Early recognition of AFE and immediate interventions are the keys to decreasing maternal morbidity and mortality (Oi et al., 2014; SMFM et al., 2016). Treatments including cardiopulmonary resuscitation, hemodynamic stabilization, volume replacement, and emergent cesarean play a critical role in survival. Nurses can assist with notifying and organizing the multidisciplinary team, blood draws for laboratory tests, line placement, and hemodynamic and blood loss assessments, along with supporting the woman and her family and keeping them informed about what is going on (Table 5). When AFE is suspected, involvement of the anesthesia team and nurses providing symptom-specific interventions are important first steps. Clear, concise communication with the obstetrician about the gravity of the clinical situation is essential to promote immediate return to the bedside if he or she has left to attend to other responsibilities. As the signs and symptoms worsen, notifying the blood bank, requesting consults from specialized gynecological surgeons and hematologists help to provide a multifaceted approach. Prompt interventions are imperative to survival and decrease morbidity. Implementing drills, simulations, and protocols with the multidisciplinary team may be useful in promoting a positive outcome. After an acute emergency such as AFE, a debriefing session to evaluate the team response, including what went well and opportunities for improvement is suggested. The patient and her family members may offer valuable feedback. When the outcome was not ideal, some team members may benefit from stress and/or grief counseling. ✣

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